Published in final edited form as:

Psychol Assess. 2011 June ; 23(2): 427–436. doi:10.1037/a0022112.

Does Response Variability Predict Distractibility among Adults with Attention-Deficit/Hyperactivity Disorder?

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Abstract

Increased intra-individual variability in response time (RTSD) has been observed reliably in ADHD and is often used as a measure of inattention. RTSD is assumed to reflect attentional lapses and distractibility, though evidence for the validity of this connection is lacking. We assessed whether RTSD is an indicator of inattention by comparing RTSD on the stop-signal task (SST) to performance on the Delayed Oculomotor Response (DOR) Task, a measure of distractibility. Participants included 30 adults with ADHD and 28 controls. Participants completed the SST and the DOR task, which measured subjects' ability to maintain attention and avoid distraction by inhibiting reflexive saccades toward distractors. On the SST, the ADHD group was slower to inhibit than controls, indicating poorer inhibitory control in ADHD. The ADHD group also displayed slower RTs, greater RTSD, and more omission errors. On the DOR task, the ADHD group displayed more premature saccades (i.e., greater distractibility) than controls. Greater variability in RT was associated with increased distraction on the DOR task but only in ADHD participants. Results suggest that RTSD is linked to distractibility among adults with ADHD and support the use of RTSD as a valid measure of inattention in ADHD.

Keywords

ADHD; adults; variable attention; eye movements

Along with deficits in response inhibition, increased intra-individual variability in response time has been reliably observed among individuals with ADHD, leading some theorists to describe this inconsistency in responding as a hallmark of the disorder (Castellanos & Tannock, 2002). Whereas deficits in response inhibition are often linked to hyperactive/impulsive symptoms of ADHD, variability in response time (RTSD) is frequently assumed to reflect inattention. More specifically, the greater RTSD values observed among

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individuals with ADHD on tasks such as the stop-signal task are thought to be a result of lapses in attentional control or distractibility. Little work has been done to evaluate the validity of this theorized connection, however. Thus, the purpose of the present study was to assess whether RTSD is a valid indicator of inattention by comparing RTSD on the stop-signal task to performance on a direct measure of distractibility among adults with ADHD and controls.

Attention-deficit/hyperactivity disorder (ADHD) is described as a heterogeneous disorder characterized by impairing symptoms in the domains of inattention, hyperactivity/impulsivity, or both (APA, 1994). There has been significant effort dedicated to uncovering the causes and correlates of ADHD over the years. This movement has evolved to examine increasingly sophisticated mechanisms and processes that underlie the disorder. One of the most fruitful lines of this kind of research has involved examining the basic cognitive processes that are theorized to contribute to symptoms of hyperactivity, impulsivity, and inattention. For instance, Barkley's (1997) influential model of ADHD denotes deficits in response inhibition as the principal pathology in ADHD. These basic problems in inhibition interact with other neurocognitive processes, generating downstream effects on overt behavior. The importance of inhibitory deficits in the development of ADHD has been widely and reliably supported across scores of studies in both children and adults (Carr, Nigg, & Henderson, 2006; Nigg, 2001, 2006).

Generally, studies in this area rely heavily on simple behavioral tasks designed to target specific psychological mechanisms or processes. In the area of response inhibition alone, dozens of tasks have been developed and tested with regard to their capacity to validly assess inhibitory function, as well as their utility in differentiating individuals with ADHD from controls (Nichols & Waschbusch, 2004; see also Nigg, 2001, Table 1). For example, two recent meta-analytic studies incorporating 35 independent samples (Lijffijt et al., 2005; Oosterlaan et al., 1998) have reviewed performance among children with ADHD on the stop-signal task (Logan & Cowan, 1984), a commonly used measure of behavioral response inhibition. Across these studies, a consistent pattern is that children with ADHD have more difficulty withholding a response than controls, as evidenced by a longer stop signal reaction time (SSRT).

Widespread use of these basic behavioral tasks in the study of ADHD has yielded another reliable trend in the literature. Many of these tasks, including the stop-signal task, require participants to respond quickly to some target stimulus. Because there are differences in individuals' trial-to-trial performance, response times are generally reported as averages, collapsed across trials. This average response time (RT_{mean}) provides an indicator of general information processing speed. Although RT_{mean} provides valuable information about an individual's performance, the use of an average obviates examination of intra-individual variability. Thus, a variable representing this intra-individual variability (RTSD) is typically reported along with other dependent variables tapped by basic behavioral tasks.

Individuals with ADHD tend to demonstrate greater RTSD relative to individuals without the disorder (Castellanos & Tannock, 2002; Derefinko et al., 2008; Gilden & Hancock, 2007; Nigg, 2006). This pattern of increased RTSD among individuals with ADHD has been frequently observed on other tasks requiring speeded responses in both children and adults (Castellanos, Sonuga-Barke, Scheres, Di Martino, Hyde, & Walters, 2005; Vaurio, Simmonds, & Mostofsky, 2009). For example, variability in reaction time on the Conners Continuous Performance Task (CPT) was effective in significantly differentiating adults with ADHD from comparison participants in two large-scale studies of ADHD in adulthood (Barkley, Murphy, & Fischer, 2007). When used to contrast children with ADHD from comparison peers, intra-subject variability on a variety of tasks yielded a larger effect size

than mean RT, omission errors, or inhibitory failures (Klein, Wendling, Huettner, Ruder, & Peper, 2006). Further, Flory and colleagues (2006) found increased RTSD to be the most influential mediator of story comprehension deficits among children with ADHD when a number of other executive function measures were included in the analyses. These examples illustrate the utility of intra-individual variability in differentiating individuals with ADHD from their peers.

Citing the ubiquity of the relation between increased response variability and ADHD in the literature, some theorists argue that intra-individual response variability is a defining feature of ADHD that could serve as a useful endophenotype for the disorder (Castellanos et al., 2005; Castellanos et al., 2005; Klein et al., 2006). Increased intra-individual variability has been linked to higher levels of disinhibited responding and inattention (Rommelse et al., 2007; Simmonds et al., 2007), two primary symptom domains of ADHD. Additionally, there is evidence for a genetic mechanism underlying this phenomenon, as supported by the observation that relatives of individuals with ADHD also tend to demonstrate greater variability in responding, regardless of their own ADHD diagnostic status (Bidwell, Willcutt, DeFries, & Pennington, 2007; Rommelse et al., 2007). Hence intra-individual response variability seems to hold great potential toward building our understanding of the developmental processes involved in the behavioral symptoms of ADHD.

Researchers have recently focused on understanding what this inconsistency in responding reflects in terms of neurocognitive functioning (DiMartino et al., 2008; Johnson et al., 2007; Simmonds et al., 2007; Suskauer et al., 2008) and how inconsistent responding in ADHD may be different from response variability in the general population (Gilden & Hancock, 2008). A number of possible explanations have been proposed, including problems in motor timing (Toplak, Dockstader, & Tannock, 2006), state regulation (Uebel et al., 2010), the default mode network (Sonuga-Barke & Castellanos, 2007), and attention. In a common explanation, inconsistent response time in ADHD has been described as a marker of inattention. In this view, the increased variability in responding and corresponding attention problems partly appear to reflect endogenous processes, such as low-frequency oscillations in neuronal activity in the default mode network that lead to lapses in attention (Castellanos et al., 2005; DiMartino et al., 2008; Vaurio et al., 2009).

Contextual factors, such as distracting stimuli in the environment, can also contribute to increased intra-individual response variability. When attending to a target stimulus in the environment, individuals must select the relevant information on which to focus (i.e., attend to the target) while simultaneously ignoring irrelevant information (Godijn & Theeuwes, 2003). When attending intentionally to a target, individuals are exercising endogenous, or internal, top-down control over attention and gaze. If, however, another object in the environment pulls attention away from the target, the resulting shift in attention and eye movement is described as exogenously-driven. Such bottom-up, automatic, stimulus-driven saccades override voluntary attentional control, resulting in lapses in attention to the target. One's propensity to commit exogenous saccades toward these non-target stimuli (i.e., distractors) rather than a target stimulus may thus be thought of as a measure of distractibility. Such occasional, momentary lapses in attention could result in greater variability in response time across trials on speeded response tasks like the SST. On trials when these lapses in attention occur, participants may miss cues to initiate responding, yielding longer RT values for those trials. This would not only increase the overall mean reaction time across trials, but would expand the range of reaction times thus resulting in greater RTSD. If individuals with ADHD have more of these momentary lapses in attention, then we would expect the ADHD group to demonstrate greater mean RT and greater RTSD than peers without ADHD.

While the link between RTSD and inattention carries intuitive appeal, the validity of this relation has yet to be assessed systematically. One strategy for evaluating the validity of RTSD as a measure of inattention would be to compare RTSD values to a direct measure of attentional functioning. The delayed oculomotor response (DOR) task measures an individual's ability to intentionally inhibit reflexive saccades toward the sudden appearance of a distracting visual stimulus on a screen. During the DOR task, participants are instructed to maintain focus on a fixation point while a bright target stimulus is presented peripherally. Typically, the sudden appearance of such a distractor would elicit a reflexive saccade toward the stimulus (Peterson, Kramer, & Irwin, 2004). However, in the DOR task participants are instructed to "delay" looking at this stimulus, and instead maintain their gaze on the fixation point until it disappears. When the target stimulus disappears, participants are instructed to look at the location on the screen where the target was. In this way, participants must inhibit an exogenous saccade when the target is presented and perform a delayed endogenous saccade once the target disappears. The primary measure of interest for the DOR task is the number of premature saccades participants commit. Premature saccades are defined as eye movements to the target stimulus before it disappears and represent failures in participants' ability to withhold or inhibit reflexive saccades. Thus, the DOR task serves as a measure of one's susceptibility to distractibility, a component of inattention.

The DOR task has been used successfully as a measure of distractibility (i.e., inhibitory control in selective attention) among groups who have been shown to have deficits in attentional control, such as individuals with schizophrenia (Ross, Heinlein, Zerbe, & Radant, 2005), children and adults with ADHD (Ross, Harris, Olincy, & Radant, 2000; Ross, Hommer, Breiger, Varley, & Radant, 1994), older adults (Gottlob, Fillmore, & Abroms, 2007), and those under the influence of alcohol (Abroms et al., 2006). In each of these groups, DOR task performance was characterized by increased inhibitory failures (i.e., looking at the distracting target stimulus prematurely) compared with healthy controls. This research supports the use of DOR task performance, particularly the proportion of premature saccades, as an indicator of distractibility among clinical groups.

The Current Study

The purpose of the current study is to assess whether RTSD is a valid indicator of inattention by comparing RTSD to performance on a direct measure of distractibility among adults with ADHD and controls. First, we predicted that participants with ADHD would show increased SSRT and RTSD values relative to controls on the stop-signal task, consistent with the substantial literature demonstrating these findings among children and, to a lesser degree, adults. Second, we predicted that individuals with ADHD, as contrasted with controls, would perform more premature eye movements to the target on a measure of distractibility. Next, we predicted that RTSD would be positively related to distractibility. Finally, the specificity of the relation between RTSD and distractibility was examined by evaluating the relations between distractibility and other performance outcome variables from the stop task (i.e., RTmean, SSRT). We predicted that distractibility would not be significantly related to RTmean, a measure of overall response speed, or SSRT, a measure of basic inhibitory functioning, reflecting a specific relation between distractibility and RTSD.

Method

Participants

Participants included 30 individuals with ADHD (17 men and 13 women; M age = 21.1 years, $SD = 1.7$) and 28 individuals with no history of ADHD (13 men and 14 women; M age = 22.0 years, $SD = 1.7$). Participants were recruited through advertisements (i.e., newspaper ads and posters) seeking adults for a study of neurological and motor

functioning. Participation was limited to individuals who were between the ages of 19 and 30 and had no uncorrected vision problems. Individuals with current psychiatric diagnoses other than ADHD were not invited to participate.

Participants in the ADHD group were recruited through advertisements specifically seeking adults with diagnosed ADHD. To ensure that members of the ADHD group were actively experiencing ADHD symptoms, only the potential participants who were currently prescribed medication for ADHD were invited to participate. Members of the ADHD group reported several different prescriptions, including Adderall ($n = 7$), AdderallXR ($n = 11$), AdderallXR and Adderall ($n = 5$), Concerta ($n = 2$), Dexedrine ($n = 2$), Daytrana ($n = 1$), and Ritalin ($n = 2$). Furthermore, these individuals were asked to provide informed consent for the access of medical records for the purpose of confirming the diagnosis. The ADHD group only included individuals whose diagnosis could be confirmed through medical records. To eliminate potential confounds associated with medication usage, participants were asked to discontinue the use of their medication for 24 hours prior to the study. Participants confirmed compliance with this request at the beginning of each session.

In addition to medical records, ADHD diagnosis was confirmed by meeting symptoms-based criteria on at least two of three ADHD scales, including the ADD/H Adolescent Self-Report Scale—Short Form (AASRS; Robin & Vandermay, 1996), the Conners Adult ADHD Rating Scale—Long Form (CAARS—S:L; Conners, Erhardt, & Sparrow, 1999), and an ADHD Symptom Checklist of 12 ADHD symptoms that serve as diagnostic criteria according to the Diagnostic and Statistical Manual of Mental Disorders (4th ed. [DSM-IV]; American Psychiatric Association, 1994). All diagnoses were confirmed by a licensed clinical psychologist with over 30 years of experience in diagnosing ADHD. This method of diagnosis confirmation has been successfully used by this research group in other studies (e.g., Weafer, Camarillo, Fillmore, Milich, & Marcinski, 2008; Weafer, Fillmore, & Milich, 2008). The AASRS—Short Form assesses symptoms experienced within the last month; this provided confirmation that participants were currently experiencing ADHD symptoms. The utilized cutoff score for this scale was the recommended diagnostic criterion of 10 or higher (Robin & Vandermay, 1996). The current study used the total ADHD symptoms subscale on the CAARS—S: L, which is based on well-established DSM-IV criteria of ADHD. The items on this scale assess the presence of ADHD symptoms throughout adulthood. For the current study, the diagnostic threshold criterion for the CAARS—S: L was a T score greater than 65 on the ADHD symptoms scale. Both scales have been shown to have sufficient specificity and sensitivity in identifying individuals with ADHD (Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999; Robin & Vandermay, 1996). Furthermore, these scales were used because they focus on ADHD symptoms as they are experienced by adults. The ADHD Symptom Checklist was created using DSM-IV items that loaded highly on the ADHD symptoms factor on the Young ADHD Questionnaire-Self-Report (Young, 2004). The scale emphasizes adult ADHD symptoms and includes six symptoms of inattention and six symptoms of hyperactivity. The respondent rates the frequency of symptom occurrence as *not at all*, *sometimes*, *often*, and *very often*. A symptom rated as occurring *often* or *very often* was counted and a total symptom count of either four or more on a single scale or seven or more on the full measure was used as a diagnostic criterion, consistent with recommendations by Barkley and colleagues (2007, p.126-127). It is important to note that the field lacks consensus on the most appropriate strategy for diagnosing ADHD in adulthood, with research suggesting that even the conventional DSM-IV criteria may not be appropriate for adults with ADHD (Barkley, Murphy, & Fischer, 2007). Hence, the multi-step classification strategy used to assign participants to the ADHD group in the current study was developed to obtain a convergence of evidence from multiple sources of information in order to confirm diagnostic status.

All participants were screened using health questionnaires, a medical history interview, and a computerized version of the Structured Clinical Interview for DSM Disorders. These measures assessed participants' current or past medical disorders, including serious physical disease, impaired cardiovascular functioning, chronic obstructive pulmonary disease, seizure, head trauma, CNS tumors, and psychiatric disorders. Participants in the control group reported past diagnosis of depression and/or anxiety ($n = 4$), head trauma ($n = 1$), and alcohol abuse ($n = 1$). One participant from the comparison group was excluded due to prior diagnosis of bipolar disorder. Those in the ADHD group reported past diagnosis of depression and/or anxiety ($n = 5$), head trauma ($n = 2$), alcohol abuse ($n = 2$), and a learning disability ($n = 1$). No participants met diagnostic criteria for any psychiatric disorders at the time of testing.

Participants completed the 30-item Barratt Impulsiveness Scale (BIS; Patton, Stanford, & Barratt, 1995) as an additional measure of impulsivity. The BIS served to further validate group classification. Impulsivity is a core characteristic associated with ADHD; the scale was administered to verify difference in this dimension between the ADHD and control groups. This questionnaire measures impulsivity through items such as "I act on impulse" and "I consider myself always careful". Participants indicate how frequently each statement applies to them on a 4-point Likert scale (*never, occasionally, often, almost always*). Possible score totals range from 30 to 120, with higher scores indicating greater total levels of impulsiveness. Group comparisons on the BIS confirmed the expected differences in impulsivity, with the ADHD group reporting greater levels of impulsivity than the control group, $t(55) = 6.47$, $p < .001$, $d = 1.74$. Table 1 compares groups in diagnostic criteria.

Tasks

Stop-signal Task—This task was used as a measure of behavioral inhibition. The task requires participants to press a button when a stimulus (go signal) appears on the screen, but to withhold responding when a stop signal tone is presented. The go signals—white circles measuring 8 mm in diameter—were presented individually. Each trial began with a 1000 msec presentation of a plus sign (+) in the middle of the computer display. This served both as a location for participants to fixate their attention and as an indication that a trial was about to begin. As soon as the plus sign disappeared, a circle appeared in one of four positions: far right (12 cm from center), middle right (6.5 cm from center), far left (12 cm from center), middle left (6.5 cm from center). Participants were required to press the forward slash key (/) on a standard computer keyboard as soon as they detected a circle on the right or the period key (.) if the circle is on the left, using their middle and index fingers, respectively. The circle was present on the screen for 1 second. A blank screen appeared for 1500 ms before the start of the next trial. The complete task involved 128 trials, with each of the four stimulus positions presented an equal number of times (32 times). A stop signal tone occurred on 32 trials (i.e., 25% of the time), equally distributed among circle positions. The stop signal was a 500 ms 900 Hz tone generated by the computer at a comfortable listening level. Participants were instructed to withhold (i.e., inhibit) their response when a stop signal is presented. Stop signals were presented eight times at each of four stimulus onset asynchronies (SOAs; i.e., delays: 50, 150, 250, and 350 ms) with respect to the onset of the circle presentation. The order of circle locations, stop signal presentation, and delays was random. A test required approximately 8 minutes to complete.

The primary dependent variable for the stop task is the SSRT, which represents an estimate of the time it takes an individual to withhold a response. SSRT is calculated here according to Logan (1994), consistent with Oosterlaan et al. (1998) and is averaged across diagnostic subgroup. The intrasubject variability on go trials (RTSD) was evaluated as the measure of inattention (Klein, Wendling, Huettner, Ruder, & Peper, 2006). Omission errors were also

considered as an indicator of inattention, as greater variability in responding can result in missed responses on go-trials. Reaction time for go trials was measured as an indicator of overall response speed. This variable was also used to calculate the potential trade-off between speed and accuracy (i.e., successful inhibitions) for each group. Anticipatory responses that took place within 100 ms of the stimulus presentation were excluded; responses committed more than 1000 ms following the stimulus to account for trials where participants may have waited to hear whether a stop signal was presented before responding. Finally, choice errors (i.e., hitting the incorrect button on a go trial) were also measured.

Delayed oculomotor response (DOR) task—This task measured the subject's ability to maintain attention and avoid distraction by inhibiting the tendency to make a reflexive saccade toward the sudden appearance of a visual stimulus on a computer screen (Ross et al., 1994;2000;2005). A chin rest was used to stabilize head movement and maintain a constant eye-to-screen distance of 73.7 cm throughout the task. Saccades were recorded using a Model 504 Eye Tracking System (Applied Science Laboratory, Boston, MA, USA). Eye locations were sampled at 60 Hz and given X/Y coordinates.

Participants were seated in a darkened room and instructed to maintain focus on a fixation point. While participants attended to the fixation point, a bright target stimulus was presented in the periphery. The onset of such a stimulus in this context normally causes a saccade to be reflexively executed toward the stimulus (Peterson et al., 2004; Theeuwes et al., 1999). However, in the DOR task, subjects are instructed to “delay” looking at this stimulus (i.e., intentionally inhibit the reflexive saccade), and instead maintain their gaze on the fixation point until it disappears. This instruction allowed differentiation among premature saccades (i.e., occurred after target presentation but before fixation point offset, during the delay), late saccades (i.e., occurred after the next fixation point appeared, beginning the next trial), and valid saccades (occurred after the delay, during the 1,000 ms response period).

A trial began with the presentation of a white fixation point (+) with a luminance of 39.6 lux presented against a black background. Participants were instructed to fixate on this point. After 1,500 ms, the target stimulus (a white circle) briefly appeared for 100 ms to the left or to the right of the fixation point. The fixation point then remained alone on the screen for a random “wait” interval (800, 1,000, and 1,200 ms). Participants were to withhold any saccade to the target. After the wait interval, the fixation extinguished and the display was blank for 1,000 ms; the disappearance of the fixation point was the signal for participants to then make a saccade to the location in which the target had appeared as quickly as possible.

A test consisted of 96 trials. Fixation points and targets were presented in five locations that were separated from each other by 4.1° of visual angle. The five positions were located horizontally across the center of the screen. Each trial began with the presentation of the fixation point at the target location of the preceding trial. Each fixation point and target location was presented on an equal number of trials during a test (24 trials at each angle, 12 in each direction). The three different wait intervals occurred in an equal number of trials (32 trials each). The target locations and wait intervals were presented in a random, unpredictable sequence. A test required 7 minutes to complete.

The task required one primary saccade per trial. This was the first saccade of each trial that occurred after target stimulus onset and covered at least half the distance to the target location for that particular trial. Thus, 96 primary saccades were required for a test. Primary saccades were categorized based on when they occurred. They were classified as either premature, valid, or late. The two primary measures of this task were the number of premature saccades, indicating distraction, and the response time of valid saccades (saccadic

RT). Saccadic RT referred to the time in milliseconds required from fixation offset to the completion of the saccade. Lower RT values represented faster saccades. Other dependent variables included saccadic accuracy (i.e., looking in the correct direction when appropriate) and number of omission errors (i.e., not looking at the target when directed to do so). Accuracy refers to the angular discrepancy between the target position and the landing point of the saccade when the target is no longer present (a measure of visual working memory). The difference between these two locations was measured in terms of degree of absolute deviation, and greater deviation scores indicated poorer accuracy of saccades. Previous findings have indicated that while ADHD children (Ross et al., 1994) and ADHD adults (Ross et al., 2000) have an increased rate of premature saccades compared to controls, they have similar accuracy of proper saccades. However, schizophrenic adults show both a higher rate of premature saccades and decreased saccade accuracy, indicating a more diffuse deficit in visual working memory (Ross et al., 2000).

Procedure

This study took place in a laboratory setting in the university's Department of Psychology. These tasks were administered as part of a larger testing battery that included other measures of cognitive functioning. Participants first attended an individual familiarization session in which they became acquainted with the eye tracking tasks and provided background information. After providing informed consent, participants were interviewed and completed questionnaires concerning their health status, drug and alcohol use, impulsivity, and demographic characteristics. The experimenter then administered the Kaufman Brief Intelligence Scale (K-BIT) in order to assess IQ. Participants in the ADHD group provided a signed release of their medical records and were interviewed regarding any medications currently prescribed for the disorder. All participants completed the ADHD scales. Participants then completed a single session consisting of a number of cognitive tasks to ensure that they understood the procedures. Following the task completion, a testing session was scheduled. The testing session was separated from the familiarization session by a minimum of 24 hours.

The testing session began with the participant completing preliminary questionnaires (e.g., verification that participants had not taken any medication). Upon completion of these questionnaires, participants were reminded of the requirements of the tasks. The tasks were then administered to the participants. To avoid fatigue effects, participants were allowed breaks as needed between tasks. After the testing session concluded the participants were debriefed and compensated approximately \$50 per session.

Results

Potential Covariates

A chi-square analysis found that gender make-up was independent of group, $\chi^2(1, n = 57) = .41, p = .52$. Moreover, no significant gender differences were found in task performance ($ps > .14$). Thus, gender was not included in any analyses as a factor or covariate. Groups differed significantly on KBIT matrices (i.e., nonverbal IQ), $t(55) = 2.24, p = .029, d = .60$, and a marginally significant difference was observed for age, $t(55) = 1.99, p = .052, d = .54$. Results did not differ when age, lifetime history of comorbid psychopathology, or verbal and nonverbal IQ scores on the KBIT were included as covariates in the analyses. Therefore, analyses are reported without the inclusion of these covariates. Additionally, the pattern of results did not change when individuals with a history of head injury ($n = 3$) were excluded. Thus, these individuals were retained in analyses reported here.

Stop-signal Task Performance

Group differences on the stop-signal task were compared using independent-samples *t* tests (see Table 2). Compared with controls, the ADHD group required more time to inhibit their responses to stop-signals, indicating poorer inhibitory control, $t(55) = 2.37, p = .02, d = .64$. Also, as expected, those in the ADHD group displayed significantly slower responses to go signals (RTmean), $t(55) = 2.73, p = .01, d = .74$, and greater variability in their RT (RTSD), $t(55) = 3.42, p = .001, d = .92$. More omission errors were observed in the ADHD group, $t(55) = 2.76, p = .01, d = .74$, however, no significant group difference in choice response errors was observed, $t(55) = 1.26, p = .21$. These results document that the adults with ADHD in the current study exhibited the typical pattern of performance for ADHD samples on a stop-signal task, namely, slower and more variable reaction times, more omission errors, and greater difficulty inhibiting responses, as measured using SSRT.

DOR Task Performance

The number of premature saccades was compared between groups using a two-sample *t*-test (see Table 2). The findings showed that, compared with the control group ($M = 9.22, SD = 6.80$), the ADHD group ($M = 21.57, SD = 13.85$) displayed significantly more premature saccades during the test, indicating greater distractibility among individuals with ADHD, $t(55) = 4.20, p < .001, d = 1.13$. No significant group differences were observed on the other measures of the DOR task (i.e., late saccades, accuracy of valid saccades, saccadic RT, and omission errors).

Relation between RTSD and Premature Saccades

Having established that the adults with ADHD demonstrated the expected patterns of problems on both the stop signal and DOR tasks, the next step in the analyses involved examining how RT variability on the stop-signal task (RTSD scores) related to number of premature saccades on the DOR task. To test this relation, a hierarchical regression analysis was conducted with premature saccades set as the dependent variable; group was entered as a predictor in the first step to determine whether group status (i.e., ADHD vs. control) accounted for a significant proportion of the variance in premature saccades. RTSD was entered in the second step, to determine whether RTSD accounted for a significant proportion of the variance in premature saccades beyond group status. Finally, a group $\times$ RTSD interaction term was entered in the third step to determine whether there were unique relations between RTSD and premature saccades for each group. Results of the regression are presented in Table 3. The regression revealed a significant group $\times$ RTSD interaction, $F(3, 53) = 16.64, p < .001$, indicating that the relation between RTSD and number of premature saccades differed by group. To understand the nature of this interaction, bivariate correlations between RTSD and premature saccades were calculated separately for each group. Results from these analyses indicate that the relation between RTSD and number of premature saccades was significant for the ADHD group ($r = .61, p < .001$), but not for the control group ($r = .24, p = .24$).

Similar regression analyses were undertaken to examine the degree to which RT variability on the stop-signal task predicted other dependent measures from the DOR task, including frequency of late saccades, saccadic RT, accuracy of saccades, and omission errors (see Table 3). There were no significant effects for the frequency of late saccades, the speed of valid saccades, or omission errors on the DOR task. That is, there were no main effects for group or RTSD in predicting these outcome measures on the DOR task, nor were there significant group $\times$ RTSD interaction effects for these variables. For the measure of the accuracy of the saccades, there was a marginally significant group $\times$ RTSD interaction, $F(3, 53) = 2.34, p = .08$. Examination of the bivariate correlations between RTSD and accuracy of saccades for each group revealed a marginally significant correlation for the ADHD

group ($r = .32, p = .08$) that was in the opposite direction of the correlation observed for the control group ($r = -.34, p = .08$).

RTmean has been identified in the literature as a potential confound to be controlled for in the relation between RTSD and distractibility. Also, given that distractibility reflects problems in inhibiting premature saccades toward a distracting target, it may be that SSRT—a measure of motoric inhibitory functioning—would be related to distractibility. Further, zero-order correlations indicate that number of omission errors on the stop-signal task was significantly correlated with RTSD (Control, $r = .40, p < .05$; ADHD, $r = .45, p < .05$) in the current sample (see Table 4). Thus, to test the relative influence of these variables in predicting distractibility, RTSD, RTmean, SSRT, and stop-signal task omission errors were entered simultaneously into a regression equation to control for the variance shared among these variables. Results indicated that only RTSD remained a significant predictor in this analysis when the ADHD group was considered alone, $\beta = .62, t(25) = 2.11, p < .05, r_{\text{semipartial}} = .33$, and when the full sample was included in the analysis, $\beta = .62, t(52) = 2.66, p = .01, r_{\text{semipartial}} = .29$. This suggests that RTSD provides a significant increment in variance for premature saccades over RTmean, SSRT, and omission errors on the stop-signal task.

Discussion

The major aim of this study was to determine whether RTSD on the stop-signal task is a valid measure of inattention and, more specifically, distractibility. Our findings supported this notion in that task performance on the DOR was strongly related to intraindividual variability of the stop-signal task. This finding provides an important step in developing the construct validity of RTSD. Prior studies have demonstrated the utility of RTSD in terms of differentiating between individuals with ADHD and unaffected peers and have advanced an assumption that this variable reflected problems in attention (Castellanos & Tannock, 2002). Although research has started to examine the relation between RTSD and some endogenous sources of inattention, including low frequency oscillations in neural functioning (Castellanos et al., 2005; DiMartino et al., 2008), we know of no work that has examined the validity of RTSD as an indicator of one's sensitivity to distraction by external stimuli. The results of the current study provide empirical support for this assumption and offer validity to discussion of RTSD as an indicator of attentional lapses and distractibility.

In the ADHD group, RTSD appeared to be uniquely related to number of premature saccades on the DOR task, as RTSD was not significantly related to the frequency of late saccades, speed of valid saccades, or accuracy of saccades. In other words, the processes underlying variable and inconsistent response times on the stop task are related to one's propensity to be distracted by external stimuli, but not to other factors like efficiency of visual processing, the integrity of the visual system, or overall information processing speed or accuracy. This pattern of findings suggests that RTSD does index distractibility and supports the continued use of RTSD as an indicator of inattention in ADHD.

There was an interaction between group and RTSD in predicting premature saccades on the DOR task, whereby RTSD only predicted premature saccades in the ADHD group. One explanation for this observed interaction may simply be that individuals in the comparison group demonstrated a relatively restricted range of premature saccades on the DOR task. It may be that increased variability in RT predicts distractibility in the general population, but the association is most evident in groups who display greater individual differences in distraction, such as those with ADHD. Notably, despite not reaching statistical significance, the correlation of .24 between RTSD and number of premature saccades in the control group represents a medium effect (Cohen, 1988). Future work with larger samples should explore

this relation in non-ADHD samples to determine whether the relation between RTSD and premature saccades is specific to ADHD or universal and manifested more strongly among individuals with attention problems.

One concern about the use of RTSD as a unique outcome variable is the substantial correlation between RTSD and RTmean. Researchers have suggested that speed and variability of responding may be correlated at a point to which they provide redundant information (e.g., $r = .92$; Wagenmakers & Brown, 2007). In the current study, there was a large amount of overlap between RTSD and RTmean. However, results support the use of RTSD over RTmean as a criterion measure of attentional control in that RTSD accounted for all of the shared variance between RTmean and premature saccades and a significant proportion of unique variance (i.e., not related to RTmean). In addition, a number of studies have found a significant relationship between RTSD, but not RTmean, and ADHD diagnosis (Nigg, 1999; Jennings, van der Molen, Pelham, Debski, & Hoza, 1997; Rucklidge & Tannock, 2002). Taken together, these findings suggest that the concern over redundancy between RTmean and RTSD is a substantive one, but RTSD appears to be a more sensitive predictor of attention problems than RTmean.

Other variables from the stop-signal task were identified that may have related to premature saccades on the DOR task. First, SSRT was considered a potential predictor of premature saccades given that both variables reflect aspects of inhibitory functioning. Additionally, omission errors on the stop task, which have also been described as an index of inattention, were evaluated as a potential predictor of premature saccades. However, when these variables were regressed on premature saccades simultaneously with RTSD, only RTSD explained a significant proportion of variance in premature saccades. This highlights the specificity of RTSD as a measure of distractibility relative to other stop task performance variables. The nonsignificant finding for SSRT may reflect a distinction between inhibitory motor control and control of attentional inhibition. The DOR task is thought to tap attentional control, whereas SSRT is understood as a measure of motor inhibition (Logan, 1994; Lijffijt, Kenemans, & Verbaten, 2005; Abroms et al., 2006). Notably, individuals with ADHD show impairment in both of these domains (Ross et al., 2000; Nigg 2001). However, results of the current study suggest that these two domains of impairment may be distinct. Thus, this study supports discussion of SSRT and RTSD as separate indicators of impairment in ADHD, with the former uniquely related to motor inhibition and the latter to distractibility.

The current study makes a number of contributions to the literature; however, results should be interpreted in light of several limitations. First, the DOR task assessed deficits of selective attention, but not other forms of inattention. Selective attention is only one component of attentional control, and future studies should consider other components of attention (e.g., focused attention; Marchetta, Hurks, De Sonnevile, Krabbendam, & Jolles, 2008). It may be useful to test the relation between stop-signal task performance and other components of attention, as this may provide a more comprehensive understanding of attentional factors affecting task performance.

Second, it is unclear how well results from the current study generalize to children and adolescents with ADHD. Some evidence suggests that children and adults with ADHD differ in task performance on the stop-signal task (Lijffijt et al., 2005). Future studies should include both children and adults with ADHD in order to better understand these performance differences. Additionally, future work should examine whether the current findings generalize to adults older than 30 years of age given that the current study focused on younger adults. A related limitation concerns the generalizability of the results to other

adults with ADHD, such as those recruited through a clinic or a group of adults with persistent ADHD followed from children.

Third, the current study did not examine the specific mechanisms underlying the relation between RTSD and premature saccades. Recent research focused on better understanding the sources of attentional lapses provide helpful guidance for how this work may proceed (Castellanos et al., 2005; DiMartino et al., 2008; Vaurio et al., 2009). Finally, the current study does not address other possible correlates or sources of intra-individual variability, such as problems in state regulation, motor timing deficits, abnormalities in the default mode network, or other forms of psychopathology associated with attention problems (e.g., anxiety, sleep disorders, head injury, and bipolar disorder; Brotman, Rooney, Skup, Pine, & Leibenluft, 2009). Future research is needed to further clarify the specificity of the relations observed in the current study between RTSD and attention problems, as well as how these relations manifest across clinically meaningful groups. The use of a clinical control group comprised of individuals who experience attentional impairment due to other forms of psychopathology would better inform our understanding of these processes and how they operate. It is also important for clinicians to recognize that RTSD should not be used as a primary diagnostic indicator for ADHD given that RTSD appears to be sensitive to attention problems from multiple etiologies.

The present research provides new and important evidence supporting the use of RTSD as a measure of inattention. Although RTSD frequently has been presumed to reflect attentional lapses and distractibility in ADHD, there have been no direct empirical tests of this assumption. Here, using a behavioral measure of distractibility, a strong relation between RTSD and premature saccades was observed, suggesting that RTSD is an effective measure of selective attention, particularly in the ADHD population. This relation appears to be specific, as RTSD was not related to other aspects of DOR task performance, and RTSD remained the lone significant predictor of premature saccades from the stop-signal task when other variables were included in the analysis. In sum, our findings lend support to claims that RTSD indexes inattention in ADHD, thus advancing our understanding of ADHD specifically and our knowledge regarding the relations among basic aspects of attention and behavior generally.

Acknowledgments

This research was supported by the National Institute on Drug Abuse grants DA021027 and DA005312.

References

- Abroms BD, Gottlob LR, Fillmore MT. Alcohol effects on inhibitory control of attention: Distinguishing between intentional and automatic mechanisms. *Psychopharmacology*. 2006; 188:324–334. [PubMed: 16953382]
- Aksan N, Kochanska G. Links between systems of inhibition from infancy to preschool years. *Child Development*. 2004; 75:1477–1490. [PubMed: 15369526]
- Aman CJ, Roberts RJ Jr, Pennington BF. A neuropsychological examination of the underlying deficit in attention deficit hyperactivity disorder: Frontal lobe versus right parietal lobe theories. *Developmental Psychology*. 1998; 34:956–969. [PubMed: 9779742]
- American Psychiatric Association. *Diagnostic and statistic manual of mental disorders*. 4th ed. Author; Washington, DC: 1994.
- Armstrong IT, Munoz DP. Attentional blink in adults with attention-deficit hyperactivity disorder. *Experimental Brain Research*. 2003; 152:243–250.
- Barkley RA. Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*. 1997; 121:65–94. [PubMed: 9000892]

- Barkley RA, Fischer M, Smallish L, Fletcher K. The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *Journal of Abnormal Psychology*. 2002; 111:279–289. [PubMed: 12003449]
- Barkley, RA.; Murphy, KR.; Fischer, M. ADHD in adults: What the science says. Guilford Press; New York: 2007.
- Bidwell LC, Willcutt EG, DeFries JC, Pennington BF. Testing for neuropsychological endophenotypes in siblings discordant for attention-deficity/hyperactivity disorder. *Biological Psychiatry*. 2007; 62:991–998. [PubMed: 17585884]
- Brotman M, Rooney M, Skup M, Pine D, Leibenluft E. Increased intrasubject variability in response time in youths with bipolar disorder and at-risk family members. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2009; 48(6):628–635. [PubMed: 19454918]
- Carr LA, Nigg JT, Henderson JM. Attentional versus motor inhibition in adults with attention-deficit/hyperactivity disorder. *Neuropsychology*. 2006; 20:430–441. [PubMed: 16846261]
- Castellanos FX, Giedd JN, Berquin PC, Walter JM, Sharp W, Tran T, et al. Quantitative brain magnetic resonance imaging in attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*. 2001; 58:239–295.
- Castellanos FX, Tannock R. Neuroscience of attention-deficit/hyperactivity disorder: The search for endophenotypes. *Nature Reviews: Neuroscience*. 2002; 3:617–628.
- Conners, CK.; Erhardt, D.; Sparrow, EP. Conners' Adult ADHD Rating Scales. Multi-Health Systems; Toronto, Canada: 1999.
- Cornblatt BA, Keilp JG. Impaired attention, genetics, and the pathophysiology of schizophrenia. *Schizophrenia Bulletin*. 1994; 20:31–46. [PubMed: 8197420]
- Derefinko KJ, Adams ZW, Milich R, Fillmore MT, Lorch EP, Lynam DR. Response style differences in the inattentive and combined subtypes of attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology*. 2008; 36:745–758. [PubMed: 18175214]
- Erhardt D, Epstein JN, Conners CK, Parker JDA, Sitarenios G. Self-ratings of ADHD symptoms in adults: II. Reliability, validity, and diagnostic sensitivity. *Journal of Attention Disorders*. 1999; 3:153–158.
- Epstein JN, Conners CK, Gill S, Erhardt D. Continuous performance test results of adults with attention deficit hyperactivity disorder. *The Clinical Neuropsychologist*. 1998; 12:155–168.
- Faraone SV, Biederman J. What is the prevalence of adult ADHD? Results of a population screen of 966 adults. *Journal of Attention Disorders*. 2005; 9:384–391. [PubMed: 16371661]
- Feifel D, Farber RH, Clementz BA, Perry W, Anllo-Vento L. Inhibitory deficits in ocular motor behavior in adults with attention-deficit/hyperactivity disorder. *Biological Psychiatry*. 2004; 56:333–339. [PubMed: 15336515]
- Fillmore MT. Drug abuse as a problem of impaired control: Current approaches and findings. *Behavioral and Cognitive Neuroscience Reviews*. 2003; 2:179–197. [PubMed: 15006292]
- Fillmore MT, Milich R, Lorch EP. Inhibitory deficits in children with attention deficit/hyperactivity disorder: Intentional versus automatic mechanisms of attention. *Development and Psychopathology*. 2009; 21:539–554. [PubMed: 19338697]
- Flory K, Milich R, Lorch EP, Hayden AN, Strange C, Welsh R. Online story comprehension among children with ADHD: Which core deficits are involved? *Journal of Abnormal Child Psychology*. 2006; 34:853–865. [PubMed: 17051434]
- Goldman-Rakic PS. Working memory dysfunction in schizophrenia. *The Journal of Neuropsychiatry and Clinical Neurosciences*. 1994; 6:348–357. [PubMed: 7841806]
- Godijn, R.; Theeuwes, J. The relationship between exogenous and endogenous saccades and attention. In: Hyona, J.; Radach, R.; Deubel, H., editors. *The minds eye: Cognitive and applied aspects of eye movement research*. Elsevier; New York, NY: 2003. p. 3-26.
- Godijn R, Theeuwes J. Ocularmotor capture and inhibition of return: Evidence for an oculomotor suppression account of IOR. *Psychological Research*. 2002; 66:234–246. [PubMed: 12466922]
- Guitton D, Buchtel HA, Douglas RM. Frontal lobe lesions in man cause difficulties in suppressing reflexive glances and in generating goal-directed saccades. *Experimental Brain Research*. 1985; 58:455–472.

- Hanisch C, Radach R, Holtkamp K, Herpertz-Dahlmann B, Konrad K. Oculomotor inhibition in children with and without attention-deficit hyperactivity disorder (ADHD). *Journal of Neural Transmission*. 2006; 113:671–684. [PubMed: 16082513]
- Jennings JR, van der Molen MW, Pelham W, Debski KB, Hoza B. Inhibition in boys with attention deficit hyperactivity disorder as indexed by heart rate changes. *Developmental Psychology*. 1997; 33:308–318. [PubMed: 9147839]
- Klein RM. Inhibition of return. *Trends in Cognitive Science*. 2000; 4:138–147.
- Klein CH, Raschke A, Brandenbusch A. Development of pro- and antisaccades in children with attention-deficit hyperactivity disorder (ADHD) and healthy controls. *Psychophysiology*. 2003; 40:17–28. [PubMed: 12756978]
- Klein C, Wendling K, Huettner P, Ruder H, Peper M. Intra-subject variability in Attention-Deficit Hyperactivity Disorder. *Biological Psychiatry*. 2006; 60:1088–1097. [PubMed: 16806097]
- Leth-Steensen C, Elbaz ZK, Douglas VI. Mean response times, variability and skew in the responding of ADHD children: A response time distributional approach. *Acta Psychologica*. 2000; 104:167–190. [PubMed: 10900704]
- Lijffijt M, Kenemans L, Verbaten MN, van Engeland H. A meta-analytic review of stopping performance in attention-deficit/hyperactivity disorder: Deficient inhibitory motor control? *Journal of Abnormal Psychology*. 2005; 114:216–222. [PubMed: 15869352]
- Logan, GD. On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In: Dagenbach, D.; Carr, TH., editors. *Inhibitory processes in attention, memory, and language*. Academic Press; San Diego, CA: 1994. p. 189-239.
- Lorch, EP.; Berthiaume, KS.; Milich, R.; van den Broek, P. Story comprehension impairments in children with attention-deficit/hyperactivity disorder. In: Cain, K.; Oakhill, J., editors. *Children's comprehension problems in oral and written language: A cognitive perspective*. Guilford Press; New York: 2007. p. 128-155.
- Lovejoy DW, Ball JD, Keats M, Stutts ML, Spain EH, Janda L, Janusz J. Neuropsychological performance of adults with attention deficit hyperactivity disorder (ADHD): Diagnostic classification estimates for measures of frontal lobe /executive functioning. *Journal of the International Neuropsychological Society*. 1999; 5:222–233. [PubMed: 10217922]
- Luna B, Garver KE, Urban TA, Lazar NA, Sweeney JA. Maturation of cognitive processes from late childhood to adulthood. *Child Development*. 2004; 75:1357–1372. [PubMed: 15369519]
- Marchetta NDJ, Hurks PPM, De Sonneville LMJ, Krabbendam L, Jolles J. Sustained and focused attention deficits in adult ADHD. *Journal of Attention Disorders*. 2008; 11:664–676. [PubMed: 17712171]
- Marzi, CA. Neuropsychology of attention. In: Denes, G.; Pizzamiglio, L., editors. *Handbook of clinical and experimental neuropsychology*. Psychology Press; Hove, East Sussex, UK: 1999. p. 509-524.
- Mohler CW, Wurtz RH. Role of striate cortex and superior colliculus in visual guidance of saccadic eye movements in monkeys. *Journal of Neurophysiology*. 1977; 40:74–94. [PubMed: 401874]
- Munoz DP, Armstrong IT, Hampton KA, Moore KD. Altered control of visual fixation and saccadic eye movements in attention-deficit hyperactivity disorder. *Journal of Neurophysiology*. 2003; 90:503–514. [PubMed: 12672781]
- Munoz DP, Broughton JR, Goldring JE, Armstrong IT. Age-related performance of human subjects on saccadic eye movement tasks. *Experimental Brain Research*. 1998; 121:391–400.
- Munoz DP, Dorris MC, Paré M, Everling S. On your mark, get set: Brainstem circuitry underlying saccadic initiation. *Canadian Journal of Physiology and Pharmacology*. 2000; 78:934–944. [PubMed: 11100942]
- Munoz DP, Istvan PJ. Lateral inhibitory interactions in the intermediate layers of the monkey superior colliculus. *Journal of Neurophysiology*. 1998; 79:1193–1209. [PubMed: 9497401]
- Munoz DP, Wurtz RH. Role of the rostral superior colliculus in active visual fixation and execution of express saccades. *Journal of Neurophysiology*. 1992; 67:1000–1002.
- Murphy KR, Barkley RA, Bush T. Executive functioning and olfactory identification in young adults with attention deficit-hyperactivity disorder. *Neuropsychology*. 2001; 15:211–220. [PubMed: 11324864]

- Nigg JT. The ADHD response-inhibition deficit as measured by the stop task: Replication with DSM-IV combined type, extension, and qualification. *Journal of Abnormal Child Psychology*. 1999; 27:393–402. [PubMed: 10582840]
- Nigg JT. Is ADHD a disinhibitory disorder? *Psychological Bulletin*. 2001; 127:571–598. [PubMed: 11548968]
- Nigg JT, Butler KM, Huang-Pollock CL, Henderson JM. Inhibitory processes in adults with persistent childhood onset ADHD. *Journal of Consulting and Clinical Psychology*. 2002; 70:153–157. [PubMed: 11860041]
- Nigg JT, Stavro G, Ettenhofer M, Hambrick DZ, Miller T, Henderson JM. Executive functions and ADHD in adults: Evidence for selective effects on ADHD symptom domains. *Journal of Abnormal Psychology*. 2005; 114:706–717. [PubMed: 16351391]
- Oosterlaan J, Sergeant JA. Inhibition in ADHD, aggressive, and anxious children: A biologically based model of child psychopathology. *Journal of Abnormal Child Psychology*. 1996; 24:19–36. [PubMed: 8833026]
- Park S, Holzman PS. Schizophrenics show spatial working memory deficits. *Archives of General Psychiatry*. 1992; 49:975–982. [PubMed: 1449384]
- Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology*. 1995; 51:768–774. [PubMed: 8778124]
- Pennington BF, Ozonoff S. Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*. 1996; 37:51–87. [PubMed: 8655658]
- Peterson MS, Kramer AF, Irwin DE. Covert shifts of attention precede involuntary eye movements. *Perception and Psychophysics*. 2004; 66:398–405. [PubMed: 15283065]
- Quay HC. Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*. 1997; 25:7–13. [PubMed: 9093895]
- Reingold EM, Stampe DM. Saccadic inhibition in voluntary and reflexive saccades. *Journal of Cognitive Neuroscience*. 2002; 14:371–388. [PubMed: 11970798]
- Ridderinkhof KR, van der Molen MW. Mental resources, processing speed, and inhibitory control: A developmental perspective. *Biological Psychology*. 1997; 45:241–261. [PubMed: 9083652]
- Robin AL, Vandermay SJ. Validation of a measure for adolescent self-report of attention deficit disorder symptoms. *Journal of Developmental and Behavioral Pediatrics*. 1996; 17:211–215. [PubMed: 8856515]
- Rommelse NNJ, Altink ME, Oosterlaan J, Beem L, Buschgens CJM, Buitelaar J, Sergeant JA. Speed, variability, and timing of motor output in ADHD: Which measures are useful for endophenotypic research? *Behavioral Genetics*. 2007; 38:121–132.
- Ross RG, Harris JG, Olincy A, Radant A. Eye movement task measures inhibition and spatial working memory in adults with schizophrenia, ADHD, and a normal comparison group. *Psychiatry Research*. 2000; 95:35–42. [PubMed: 10904121]
- Ross RG, Harris JG, Olincy A, Radant A, Adler LE, Freedman R. Familial transmission of two independent saccadic abnormalities in schizophrenia. *Schizophrenia Research*. 1998; 30:59–70. [PubMed: 9542789]
- Ross RG, Heinlein S, Zerbe GO, Radant A. Saccadic eye movement task identifies cognitive deficits in children with schizophrenia, but not in unaffected child relatives. *Journal of Child Psychology and Psychiatry*. 2005; 46:1354–1362. [PubMed: 16313436]
- Ross RG, Hommer DW, Breiger D, Varley C, Radant A. Eye movement task related to frontal lobe functioning in children with attention deficit disorder. *The Journal of the American Academy of Child and Adolescent Psychiatry*. 1994; 33:869–874.
- Rucklidge JJ, Tannock R. Neuropsychological profiles of adolescents with ADHD: Effects of reading difficulties and gender. *Journal of Child Psychology and Psychiatry*. 2002; 43:988–1003. [PubMed: 12455921]
- Schachar R, Logan GD. Impulsivity and inhibitory control in normal development and childhood psychopathology. *Developmental Psychology*. 1990; 26:710–720.
- Schachar R, Tannock R, Marriott M, Logan G. Deficient inhibitory control in attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology*. 1995; 23:411–437. [PubMed: 7560554]

- Schneider, W.; Eschman, A.; Zuccolotto, A. E-prime user's guide. Psychology Software Tools; Pittsburgh, PA: 2002.
- Sereno AB, Briand KA, Amador SC, Szapiel SV. Disruption of reflexive attention and eye movements in an individual with a collicular lesion. *Journal of Clinical and Experimental Neuropsychology*. 2006; 28:145–166. [PubMed: 16448982]
- Shimojo, S.; Hikosaka, O.; Miyauchi, S. Automatic and controlled attention detected by the line motion effect. In: Daniel, G.; Koriati, A., editors. *Attention and performance XVII: Cognitive regulation of performance: Interaction of theory and application*. MIT Press; Cambridge, MA: 1999. p. 145-163.
- Simmonds DJ, Fotedar SG, Suskauer SJ, Pekar JJ, Denckla MB, Mostofsky SM. Functional brain correlates of response time variability in children. *Neuropsychologia*. 2007; 45:2147–2157. [PubMed: 17350054]
- Sonuga-Barke EJS, Castellanos FX. Spontaneous attentional fluctuations in impaired states and pathological conditions: A neurobiological hypothesis. *Neuroscience and Biobehavioral Reviews*. 2007; 31(7):977–986. [PubMed: 17445893]
- Sparks DL. Functional properties of neurons in the monkey superior colliculus: Coupling of neuronal activity and saccade onset. *Brain Research*. 1978; 156:1–16. [PubMed: 100173]
- Suskauer SJ, Simmonds DJ, Caffo BS, Denckla MB, Pekar JJ, Mostofsky SH. fMRI of intrasubject variability in ADHD: Anomalous premotor activity with premotor compensation. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2008; 47(10):1141–1150. [PubMed: 18724253]
- Tannock R. Attention deficit hyperactivity disorder: Advances in cognitive, neurobiological, and genetic research. *Journal of Child Psychology and Psychiatry*. 1998; 39:65–99. [PubMed: 9534087]
- Taylor TL, Klein RM. On the causes and effects of inhibition of return. *Psychonomic Bulletin & Review*. 1998; 5:625–643.
- Toplak ME, Dockstader C, Tannock R. Temporal information processing in ADHD: Findings to date and new methods. *Journal of Neuroscience Methods*. 2006; 151:15–29. [PubMed: 16378641]
- Uebel H, Albrecht B, Asherson P, Borger N, Butler L, Chen W, Banaschewski T. Performance variability, impulsivity errors, and the impact of incentives as gender-independent endophenotypes for ADHD. *Journal of Child Psychology and Psychiatry*. 2010; 51(2):210–218. [PubMed: 19929943]
- Vaurio RG, Simmonds DJ, Mostofsky SH. Increased intra-individual reaction time variability in attention-deficit/hyperactivity disorder across response inhibition tasks with different cognitive demands. *Neuropsychologia*. 2009; 47:2389–2396. [PubMed: 19552927]
- Wagenmakers EJ, Brown S. On the linear relationship between the mean and the standard deviation of response time distribution. *Psychological Review*. 2007; 114:830–841. [PubMed: 17638508]
- Weafer J, Camarillo D, Fillmore MT, Milich R, Marczyński CA. Simulated driving performance of adults with ADHD: Comparisons with alcohol intoxications. *Experimental and Clinical Psychopharmacology*. 2008; 16:251–263. [PubMed: 18540785]
- Weafer J, Fillmore MT, Milich R. Increased sensitivity to the disinhibiting effects of alcohol in adults with ADHD. *Experimental and Clinical Psychopharmacology*. 2008; 17:113–121. [PubMed: 19331488]
- Weiss, M.; Hechtman, LT.; Weiss, G. ADHD in adulthood: A guide to current theory, diagnosis, and treatment. The Johns Hopkins University Press; Baltimore, MD: 1999.
- Young S. The YAQ-S and YAQ-I: The development of self and informant questionnaires reporting on current adult ADHD symptomatology, comorbid and associated problems. *Personality and Individual Differences*. 2004; 36:1211–1212.

Table 1

Demographic and Diagnostic Information by Group

	Comparison (<i>n</i> = 27)			ADHD (<i>n</i> = 30)			<i>t</i>
	<i>Mean</i>	<i>SD</i>		<i>Mean</i>	<i>SD</i>		
Demographic							
Age	22.0	1.7	21.1	1.7	1.7	2.0 ^I	
Education	15.3	1.3	15.1	1.0	0.7		
IQ: Verbal	107.1	6.9	103.2	10.8	1.6		
IQ: Nonverbal	111.2	8.8	105.6	9.8	2.2*		
Diagnostic							
CAARS							
DSM-IA	51.0	11.1	75.9	11.5	8.3***		
DSM-HI	46.4	9.7	64.8	14.8	5.5***		
DSM-Tot	49.0	11.4	73.7	11.8	5.6***		
Index	45.1	8.4	59.1	10.1	8.0***		
AASRS	8.1	5.6	21.4	5.8	8.8***		
DSM	0.9	1.3	5.4	2.1	9.7***		
BIS	52.6	8.4	68.5	9.9	6.5***		

Note. Group contrasts were independent sample *t* tests, where *n* = 57, *df* = 55. Age is reported in years. Education refers to years education completed. IQ: Verbal and IQ: Nonverbal refer to IQ score on the respective subtests of the Kaufman Brief Intelligence Test. CAARS refers to T scores on subscales of the Conners Adult ADHD Rating Scale—Long Form; DSM-IA is DSM-IV Inattentive Symptoms; DSM-HI is DSM-IV Hyperactive-Impulsive Symptoms; DSM-Tot is DSM-IV ADHD Symptoms Total; and Index is ADHD Index. AASRS refers to total score on the ADD/H Adolescent Self-Report Scale—Short Form. DSM refers to symptom count on the ADHD symptoms checklist. BIS refers to the total score on the Barratt Impulsiveness Scale.

^I *p* = .052
^{*} *p* < .05
^{***} *p* < .001

Table 2**Stop Signal and DOR Task Performance by Group**

Variable	Comparison (<i>n</i> = 27)	ADHD (<i>n</i> = 30)	ADHD vs. Comparison <i>t</i>
Stop-signal task			
RTmean	400.5 (60.5)	451.7 (78.7)	2.73**
RTSD	94.6 (23.9)	120.4 (31.9)	3.42**
SSRT	229.3 (50.9)	267.4 (68.1)	2.37*
Inhibitory failures (%)	62.0 (20.8)	58.2 (22.9)	.66
Omission errors	.74 (1.1)	3.1 (4.4)	2.76**
Choice errors	.52 (.80)	1.0 (1.8)	1.26
DOR task			
Premature saccades	9.2 (6.8)	21.6 (13.9)	4.20***
Late saccades	2.3 (3.9)	3.3 (3.3)	1.05
Saccadic RT	418.2 (73.1)	434.9 (85.1)	.79
Accuracy	1.6 (.40)	1.7 (.56)	.77
Omission errors	.72 (1.3)	1.1 (1.1)	1.34

Note: Group contrasts were independent samples *t* tests, where *n* = 57, *df* = 55.

*
p < .05

**
p < .01

Table 3

Results from Hierarchical Regressions of RTSD on DOR Task Outcomes

Outcome variable		Step 1	Group (ADHD vs. comparison)	df	β	SE β	ΔR^2	Comp r	ADHD r
Premature saccades	Step 1	Group		1, 55	12.3	2.9	.24***		
	Step 2	RTSD		2, 54	.20	.05	.20***		
	Step 3	Group $\times$ RTSD		3, 53	.20	.09	.04*	.24	.61***
Late saccades	Step 1	Group		1, 55	1.0	1.0	.02		
	Step 2	RTSD		2, 54	.02	.02	.02		
	Step 3	Group $\times$ RTSD		3, 53	.02	.04	.004		
Saccadic RT	Step 1	Group		1, 55	16.7	21.1	.01		
	Step 2	RTSD		2, 54	.27	.38	.01		
	Step 3	Group $\times$ RTSD		3, 53	.02	.81	.00		
Accuracy of saccades	Step 1	Group		1, 55	.10	.13	.01		
	Step 2	RTSD		2, 54	.00	.00	.01		
	Step 3	Group $\times$ RTSD		3, 53	.01	.01	.10 ^I		
Omission errors	Step 1	Group		1, 55	.41	.31	.03		
	Step 2	RTSD		2, 54	.00	.01	.00		
	Step 3	Group $\times$ RTSD		3, 53	.01	.01	.01		

* Note: $p < .05$ ** $p < .01$ *** $p < .001$ ^I $p = .08$

Table 4
Correlations among Stop Signal and DOR Task Performance Variables by Group

	1	2	3	4	5	6	7	8	9	10	11
Stop-signal task											
1. RTmean	1.00	.90***	.27	-.75***	.44*	-.20	.21	-.03	.03	-.28	-.19
2. RTSD	.85***	1.00	.30	-.62***	.40*	-.16	.24	.05	.08	-.34	-.13
3. SSRT	.25	.28	1.00	.39*	.24	.24	.23	-.01	.17	.02	.05
4. Inhibitory failures	-.76***	-.66***	.37*	1.00	-.26	.41*	.04	.07	.19	.32	.21
5. Omission errors	.51**	.45*	.03	-.42*	1.00	-.15	.24	-.08	-.08	-.10	-.01
6. Choice errors	-.18	-.10	-.10	.09	.38*	1.00	.04	.05	.15	.10	-.20
DOR task											
7. Premature saccades	.53**	.61***	.02	-.44*	.31	-.10	1.00	.10	.29	-.13	.09
8. Late saccades	.36*	.23	.00	-.29	.43*	-.13	.18	1.00	.46*	.22	.26
9. Saccadic RT	.38*	.10	.11	-.21	.38*	-.08	.06	.57**	1.00	.29	.27
10. Accuracy of saccades	.34	.32	.22	-.18	.13	.12	.33	.35 ^I	.36*	1.00	.24
11. Omission errors	-.05	.10	-.01	.02	.02	.27	.09	.08	.04	.14	1.00

Note: Values represent zero-order correlation coefficients.

*
 $p < .05$

**
 $p < .01$

 $p < .001$

^I
 $p = .06$.

Values above the diagonal are for the Control group only, and values below the diagonal are for the ADHD group only.